

Infectious Thoracic Aortitis: A Literature Review

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ABSTRACT

Infectious thoracic aortitis (IA) remains a rare disease, especially after the appearance of antibiotics. However, if left untreated it is always lethal. It usually affects patients with atherosclerotic aortic disease and/or infective endocarditis. Mycotic aneurysm is the most common form of presentation, although a few reports of nonaneurysmal infectious thoracic aortitis have also been described. Various microorganisms have been associated with infectious thoracic aortitis, most commonly *Staphylococcal*, *Enterococcus*, *Streptococcus*, and *Salmonella* species. It is extremely important to establish an early diagnosis of IA, because this condition is potentially life-threatening. However, diagnosis is frequently delayed since clinical manifestations are usually nonspecific. Antibiotherapy in combination with complete surgical excision of the infected aorta is the best choice of treatment.

Introduction

Infectious thoracic aortitis (IA) also known by bacterial, microbial, or cryptogenic aortitis, or mycotic or infected aneurysm, is a rare entity in the antibiotic era. It usually affects patients with prior atherosclerotic aorta disease and/or associated infective endocarditis (IE).^{1,2} In the pre-antibiotic era, infectious thoracic aortitis was even more frequently related with IE, occurring in 86% of cases.¹ The infection can occur in normal or atherosclerotic aortas, aneurysms included, but is far more frequent in the latter case.³ Infectious thoracic aortitis and mycotic or infected aneurysms (MA) represent 2 extremes of the same disease. If left untreated, an infected nonaneurysmal aorta (typically with atherosclerosis) will likely progress to MA. However, an MA usually develops when a preexisting aneurysm becomes secondarily infected.⁴ Mycotic aneurysms represent 2.6% of all aortic aneurysms, the thoracic aorta is the least common site of occurrence.¹ In most of the reports, IA presents with an aneurysm, and it is impossible to determine if the aneurysm was already there when the infection began.⁴ Furthermore, there are only a few cases in the literature describing IA in the absence of an aneurysm or rupture.^{3,5,6,7} Infectious thoracic aortitis has a male predominance (male:female ratio = 3:1) and the mean age at diagnosis is 65 years old. If IA occurs associated to an IE, there is no gender predominance and the mean age at diagnosis is 40 years old.¹

Pathogenesis

Aortic intima is generally highly resistant to infection, but the disruption of this barrier by atherosclerosis reduces this resistance. Infection of the aorta can arise by several mechanisms: (1) bacteremic seeding of an existing intimal injury or atherosclerotic plaque (the most common mechanism), (2) septic emboli of the aortic vasa vasorum (once the most common cause, typically in bacterial endocarditis), (3) contiguous infective focus extending to the aorta wall (rare), and (4) direct bacterial inoculation

at the time of trauma, such as a penetrating injury.^{1,3,4} Risk factors for aortic infections are obviously the same as those for atherosclerosis, but also IE, aortic trauma (such as a complication of aortic angiography), congenital aortic anomalies (such as coarctation and patent ductus arteriosus), and conditions causing impaired immunity (such as cancer, diabetes, alcoholism, and immunosuppressive therapies).^{1,8}

Microbiology

Although any microorganism can infect the aorta, certain bacteria seem to have a propensity for this location. Gram-positive bacteria such as the *Staphylococcal* species, *Enterococcus* species, and *Streptococcus pneumoniae*, are the most common, and are responsible for 60% of the infections. When IA is associated with IE, *Enterococcus* species and *Streptococcus pneumoniae* are the most common agents isolated. Gram-negative bacilli (*Salmonella* species in the majority of cases) are also frequent microorganisms causing aortic infection; however, they are more prevalent in infectious abdominal aortitis.¹

Aortic infection by *Mycobacterium tuberculosis*, an uncommon problem in developed countries, may occur as a result of the extension to the aorta wall of a contiguous infective focus (infected lymph nodes or lung lesions). *Treponema pallidum* is, currently, an exceedingly rare etiology.

Aortic infection by unusual bacteria and nonbacterial microorganisms (such as fungi) is extremely rare; however its frequency is increasing in immunodepressive patients.²

Clinical Presentation

Clinical manifestations are often nonspecific, depending upon the site of infection and aneurysm formation. Fever (75%), thoracic and dorsal pain (60%), abdominal pain (20%), and chills (16%) are the most frequent symptoms.¹ Patients with no aneurysm formation are likely to be less symptomatic.⁴ Mycotic aneurysms can cause compressive

symptoms, such as dysphagia, dyspnea, hoarseness, cough, and superior vena cava compression syndrome.¹ Possible complications of IA include aneurysm formation with subsequent rupture and bleeding, aortic thrombosis with distal embolization (cerebral, visceral, or limb), aortic dissection, septic embolization, aortic insufficiency, and acute coronary syndromes caused by coronary involvement.²

Diagnosis

It is extremely important to establish an early diagnosis of IA, because this condition is associated with a high rate of aortic rupture and mortality if left untreated.² However, diagnosis is frequently delayed since clinical manifestations are usually nonspecific or the first symptoms often result from expansion or rupture of an aneurysm. Infectious thoracic aortitis should always be considered in elderly patients with atherosclerosis, history of thoracic, abdominal, or back pain, and vague symptoms such as nausea, vomiting, and fever.⁴

Leukocytosis and neutrophilia are present in 65% to 83% of cases.¹ Erythrocyte sedimentation rate and C-reactive protein are elevated in most of the patients.² Blood cultures are positive in 50% to 85% of the patients and a microorganism can be isolated from the excised aortic tissue in up to 76% of the patients. Pretreatment with antibiotics, the absence of an infectious focus in the aortic lumen, and the presence of anaerobic microorganisms are possible causes for negative blood and tissue cultures.¹

Since IA usually affects patients with associated IE, transesophageal echocardiogram (TEE) is frequently the first imaging modality to be performed in daily practice as it is the gold standard method to rule out IE. If negative for IE the thoracic aorta should be promptly examined for evidence of aneurysm or vegetations. The immediate proximity of the esophagus to the great vessels allows optimal imaging. Moreover, tissue Doppler echocardiography can further improve structural identification. Transesophageal echocardiogram can distinguish intramural hematoma, aneurysm, pseudoaneurysm, and dissection of the thoracic aorta. Nonetheless, if the trachea is interposed, upper ascending aorta and proximal aortic arch locations may be missed.^{1,2,9,10}

Computed tomography scan (CT) with contrast enhancement is widely available in most medical centers being considered by many the initial imaging technique of choice.^{2,4} It allows a rapid exclusion of other aortic pathologies that may sometimes resemble acute aortitis, such as aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer.² Periaortic nodularity, change in aorta size, air in the aortic wall, and saccular aneurysm, are some radiological findings that may lead to an early diagnosis.⁴ Furthermore, CT findings of periaortic density and adjacent gas collection suggest an impending rupture, even if no aneurysm is present.¹⁰ However, small vegetations and milder degrees

of inflammation or wall edema may be missed. Radiation exposure and contrast need (if optimal vascular imaging is to be obtained) are other CT limitations.^{2,4}

Magnetic resonance imaging (MRI) with gadolinium contrast enhancement is emerging as an imaging modality of choice for aortitis. It provides an entire aorta image with excellent resolution and without radiation exposure. Areas of aortitis may appear as vessel wall edema, enhancement, or wall thickening. To enhance the characterization of the blood vessel wall, some specific protocols have been created, like the "edema-weighted" technique. However MRI is not widely available and it should not be performed in patients with certain implanted devices or who are unstable (since most of the resuscitations equipment cannot enter into the scanning room) if there is another acceptable imaging alternatives.^{2,4}

Magnetic resonance imaging and multislice CT scan can precisely define disease extension and help planning surgical intervention.^{2,4}

Invasive aortography is reserved for cases in which diagnosis of acute aortic syndrome cannot be excluded by noninvasive methods. It has the disadvantage of imaging the aortic lumen only and carries the risk of rupture of a fragile aortic wall.²

Management and Treatment

There are no randomized controlled studies to guide the management of IA. Most of the authors agree that antibiotic therapy in combination with complete surgical excision of the infected aorta is the best choice of treatment. The intent of surgery is to confirm the diagnosis, control sepsis, control hemorrhage (if rupture occurred), and reconstruct the arterial vasculature.¹¹

As soon as the diagnosis is suspected, intravenous antibiotics with broad antimicrobial coverage should be initiated. If surgery is not emergent (patients without impending aortic rupture or uncontrolled sepsis), it seems reasonable to perform a course of antibiotics for 2 to 4 weeks prior to surgery to improve local surgical conditions.⁹ The antimicrobial therapy should be extended for at least 6 to 12 weeks after surgical excision and clearance of blood cultures. A longer course should be considered for immunosuppressed patients and if biochemical parameters of inflammation do not return to normal.² Some authors even recommend life-long oral antibiotics following the intravenous course, especially for microorganisms difficult to treat¹³ or after in situ prosthetic bypass.¹⁴

The standard surgical management of IA involves resection of the infected aortic segment with in situ or extra-anatomic reconstruction. There is special concern over whether the type of reconstruction might contribute to an increased incidence of graft-related complications.¹⁵ Theoretically, in situ graft replacement may have a higher rate of early graft infection, and extra-anatomic bypass is more

prone to thrombosis and need of future reconstruction.¹⁶ There have been many reports on the efficacy, safety, and durability of in situ prosthetic graft, with very low rates of early graft infection and mortality.^{14,15,16} Therefore, resection of the infected aortic segment with in situ prosthetic graft reconstruction is, currently, the preferred method of revascularization.¹⁴

In the past decade, endovascular techniques have been extended to the treatment of infected thoracic aorta aneurysms, with good results.^{14,17,18} Chung-Dann et al have made a meta-analysis of all the reports of endovascular aortic repair (EVAR) performed in patients (n = 48) with thoracic and abdominal aortic MA. Both postoperative and 12-month mortality rates were 10.4%, which is better than surgical mortality rates. Infection persisted in 22.9% of the patients after EVAR and was closely associated with poor prognosis (12-month survival rate of 39% vs 94% in patients without persistent infection). Ruptured MA and fever at the time of EVAR procedure were the only factors related to infection persistence. In patients with fever and ruptured MA, EVAR should be considered only as a temporary measure to achieve hemodynamic stability.¹⁹ Endovascular techniques need further studies and longer follow-up periods in order to better define their role in IA management.¹⁴

Prognosis

Death supervenes in all IA cases that are left untreated. However, a combination of surgical and medical therapy may lead to a survival rate of 75% to 100% before aneurysm formation,¹ and 62% after an aneurysm has formed. After aneurysm rupture, surgical mortality rates may be as high as 65%.¹⁴ With medical treatment alone, mortality rates may reach 90%.⁸ In the few EVAR procedures performed worldwide, postoperative mortality rates were 10.4%.¹⁹

Factors apparently linked to a worse prognosis are advanced age, diagnosis delay, gram-negative bacilli infection, immunodepression, thoracic location, medical treatment alone, and complication occurrence (such as rupture, embolization, or septic shock).^{1,14,19} Some authors recommend close medical follow-up that includes serial blood cultures and CT scans,¹² while others do not perform such approaches, recommending only clinical surveillance.¹⁶

Conclusion

Infectious thoracic aortitis is a rare but potentially life-threatening disorder. It should be considered in patients with atherosclerotic risk factors and in whom there is a suspicion of IE or cardiac source of embolism, and TEE should be performed as early as possible. CT or MRI is indicated when a strong suspicion of IA remains and TEE was negative and/or the entire thoracic aorta could not be visualized. Even when the diagnosis is established by TEE, we consider that CT or MRI should be performed to better define the disease and help to plan surgical treatment. Early

and complete surgical excision of the infected aorta in combination with large coverage antibiotherapy (extended for at least 6–12 weeks) is the best choice of treatment. Nevertheless, a course of antibiotics for 2 to 4 weeks prior to surgery may be attempted to improve local surgical conditions in patients without impending aortic rupture or uncontrolled sepsis. Although good results have been reported with EVAR in IA management, there is not yet enough evidence to consider it as an alternative to surgery.

References

1. Revest M, Decaux O, Cazalets C, Verohye JP, Jgo P, Grosbois B. Thoracic infectious aortitis: microbiology, pathophysiology and treatment. *Rev Med Interne*. 2007;28(2):108–115.
2. Gornik HL, Creager MA. Aortitis. *Circulation*. 2008;117(23):3039–3051.
3. Bansal RC, Ashmeik K, Razzouk AJ. An unusual case of vegetative aortitis diagnosed by transesophageal echocardiography. *J Am Soc Echocardiogr*. 2001;14(3):237–239.
4. Narang AT, Rathlev NK. Non-aneurysmal infectious aortitis: a case report. *J Emerg Med*. 2007;32(4):359–363.
5. Frank MW, Mehlman DJ, Tsai F, Lomasney JW, Joob AW. Syphilitic aortitis. *Circulation*. 1999;100(14):1582–1583.
6. Wein M, Bartel T, Kabatnik M, Sadony V, Dirsch O, Erbel R. Rapid progression of bacterial aortitis to an ascending aortic mycotic aneurysm documented by transesophageal echocardiography. *J Am Soc Echocardiogr*. 2001;14(6):646–649.
7. Parkhurst GF, Decker JP. Bacterial aortitis and mycotic aneurysm of the aorta. *Am J Pathol*. 1955;31:821–830.
8. Johnson JR, Ledgerwood AM, Lucas CE. Mycotic aneurysm. New concepts in therapy. *Arch Surg*. 1983;118:577.
9. Wein M, Bartel T, Kabatnik M, Sadony V, Dirsch O, Erbel R. Rapid progression of bacterial aortitis to an ascending aortic mycotic aneurysm documented by transesophageal echocardiography. *J Am Soc Echocardiogr*. 2001;14(6):646–649.
10. Malouf JF, Chandrasekaran K, Orszulak TA. Mycotic aneurysms of the thoracic aorta: a diagnostic challenge. *Am J Med*. 2003;115(6):489–496.
11. Mantello MT, Panaccione JL, Moriarty PE, et al. Impending rupture of nonaneurysmal bacterial aortitis: CT diagnosis. *J Comput Assist Tomogr*. 1990;14:950–953.
12. Foote EA, Postier RG, Greenfield RA, Bronze MS. Infectious aortitis. *Curr Treat Options Cardiovasc Med*. 2005;7(2):89–97.
13. Brown SL, Busuttill RW, Baker JD, et al. Bacteriologic and surgical determinants of survival in patients with mycotic aneurysms. *J Vasc Surg*. 1984;1:541.
14. Ting AC, Cheng SW, Ho P, Poon JT, Tsu JH. Surgical treatment of infected aneurysms and pseudoaneurysms of the thoracic and abdominal aorta. *Am J Surg*. 2005;189(2):150–154.
15. Oderich GS, Panneton JM, Bower TC, et al. Infected aortic aneurysms: aggressive presentation, complicated early outcome, but durable results. *J Vasc Surg*. 2001;34(5):900–908.
16. Hsu RB, Tsay YG, Wang SS, Chu SH. Surgical treatment for primary infected aneurysm of the descending thoracic aorta, abdominal aorta, and iliac arteries. *J Vasc Surg*. 2002;36(4):746–750.
17. Semba CP, Sakai T, Slonim SM, et al. Mycotic aneurysms of the thoracic aorta: repair with use of endovascular stent-grafts. *J Vasc Interv Radiol*. 1998;9(1 Pt 1):33–40.
18. Van Doorn RC, Reekers J, de Mol BA, Obertop H, Balm R. Aortoesophageal fistula secondary to mycotic thoracic aortic aneurysm: endovascular repair and transhiatal esophagectomy. *J Endovasc Ther*. 2002;9(2):212–217.
19. Kan CD, Lee HL, Yang YJ. Outcome after endovascular stent graft treatment for mycotic aortic aneurysm: a systematic review. *J Vasc Surg*. 2007;46(5):906–912.